Diagnosis and management of acute mesenteric ischemia: What you need to know

Matti Tolonen, MD, PhD *and* **Pirkka Vikatmaa, MD, PhD**, *Helsinki, Finland*

ABSTRACT: Acute mesenteric ischemia (AMI) is associated with high mortality rates. There are multiple challenges to establishing an accurate early diagnosis and providing state-of-the-art care for AMI patients. A high index of suspicion is key for early diagnosis. Once suspicion is raised, a triphasic computed tomography angiography is the essential diagnostic tool. Avoiding delays, using hybrid operating rooms and contemporary revascularization techniques for arterial occlusive AMI, can significantly improve the prognosis. Regional health care systems should be developed to direct AMI patients into centers with sufficient capabilities for providing all aspects of care at all hours. The acute care surgeon has a central role in performing laparotomies and bowel resections when needed and coordinating the management flow in close collaboration with vascular surgeons and interventional radiologists for prompt and effective revascularization. A significant share of patients with an arterial occlusive AMI can be managed by endovascular revascularization without the need for a laparotomy. There are no reliable tools for predicting transmural bowel necrosis, and individual assessment and clinical experience are very important in decision-making when choosing between laparotomy and close observation. During laparotomy, an atherosclerotic occlusion at the root of the superior mesenteric artery can be stented by using a retrograde open mesenteric or percutaneous approach, and surgical bypass is seldom needed. Using hospital-specific management pathways is very useful for the standardization of care in arterial occlusive AMI. In venous AMI, systemic anticoagulation is sufficient in most cases. In patients whose symptoms do not resolve, there are various options for endovascular and surgical revascularization. In nonocclusive mesenteric ischemia, prevention by maintaining sufficient abdominal perfusion pressure is key. High-level evidence is scarce, but with current knowledge, the prognosis of AMI patients has plenty of room for improvement. (*J Trauma Acute Care Surg.* 2025;99: 151–161. Copyright © 2025 Wolters Kluwer Health, Inc. All rights reserved.)

KEY WORDS: Emergency surgery; endovascular; bowel ischemia.

Acute mesenteric ischemia (AMI) is a surgical emergency disproportionally affecting elderly patients carrying high mortality rates. In the more common arterial occlusive forms of acute mesenteric ischemia (Ao-AMI), mortality varies between 50% and 80%.[1–5] Patients with suspected AMI need to be immediately referred to a center with sufficient capabilities for revascularization, bowel resection, and intensive care management.[6,7] Currently, many hospitals are unable to effectively revascularize Ao-AMI patients.[5,8,9] With prespecified efficient regional and in-hospital multidisciplinary pathways, the mortality can be markedly lowered.[10,11] Therefore, the management of AMI has a significant potential for quality improvement worldwide. The mainstays of management of Ao-AMI are prompt diagnosis, ensuring appropriate patient access to facilities with sufficient capabilities, immediate and efficient revascularization, and resection of bowel with transmural necrosis.[2,3,10]

ANATOMY AND PATHOPHYSIOLOGY

The three mesenteric arteries are ventral branches of the abdominal aorta and supply blood flow to the viscera. The celiac artery (CA) is the most proximal, followed by the superior mesenteric artery (SMA) and the inferior mesenteric artery. There are vast networks of collateral arteries between these vessels. The venous return from the bowel occurs through the inferior and the superior mesenteric veins and the splenic vein. All these veins terminally drain into the portal vein. At resting state, the viscera receive 10% to 20% of the cardiac output, whereas, postprandially, the requirements increase to approximately 35%.[12,13] In clinical practice, SMA occlusion is the most common cause of AMI. The CA may also occasionally require revascularization. However, revascularization of the inferior mesenteric artery is experimental and not commonly targeted in general clinical practice.[2,3]

A stenotic lesion in a single mesenteric artery is not uncommon in elderly patients. Indeed, the diagnosis of mesenteric ischemia should not solely be based on imaging studies but should be supported by a constellation of clinical symptoms that suggest the diagnosis. In chronic mesenteric ischemia, frequent symptoms comprise postprandial pain and weight loss. Stenosis over 50% in the SMA or in the CA is considered clinically significant. Chronic mesenteric ischemia may progress to thrombotic acute mesenteric ischemia (TAMI). Arterial occlusive form of AMI is typically caused by a thrombus or an embolism Embolism AMI (EAMI) of the SMA (Fig. 1). Rare causes include arterial dissections, vasculitis, and somewhat controversial median arcuate ligament syndrome, where the CA is extrinsically compressed by the median arcuate ligament, causing postprandial pain.[2,14,15]

When Ao-AMI causes acute bowel ischemia with imminent necrosis, it becomes a surgical emergency. In the vast majority of cases, the affected bowel segments follow the vascularization area of the SMA, that is, from the proximal jejunum to the transverse

Submitted: December 1, 2024, Revised: January 8, 2025, Accepted: January 8, 2025, Published online: March 20, 2025.

From the Department of Abdominal Surgery (M.T.), HUS Abdominal Center; and Department of Vascular Surgery (P.V.), Abdominal Center, Helsinki University Hospital, University of Helsinki, Helsinki, Finland.

Address for correspondence: Matti Tolonen, MD, PhD, Department of Abdominal Surgery, HUS Abdominal Center, Meilahti Tower Hospital, Haartmaninkatu 4, 00290 Helsinki, Finland; email: matti.tolonen@hus.fi.

DOI: 10.1097/TA.0000000000004585

Copyright © 2025 Wolters Kluwer Health, Inc. All rights reserved.

SHOUT
SHARE
IT

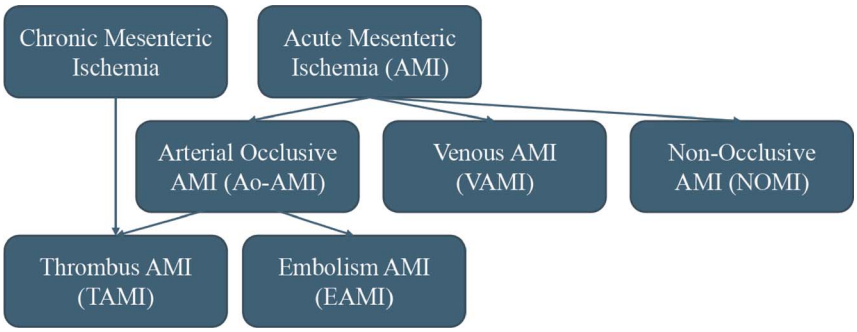

Figure 1. Classification of mesenteric ischemia.

colon. Rarely, especially combined CA and SMA occlusions may cause ischemia proximal to the jejunum.[2,3]

Venous acute mesenteric ischemia (VAMI) is less common and generally presents with a longer history and more subtle symptoms. A mesenteric venous thrombosis (MVT) in the portomesenteric venous system may cause severe venous outflow occlusion and result in VAMI. Without bowel ischemia, the diagnosis is MVT and does not warrant routine surgical consultation. Mesenteric venous thrombosis is predisposed by one or more of three factors: direct vessel wall injury, venous stasis, or prothrombotic state. Typical associated diagnoses are abdominal inflammation diseases (pancreatitis, peritonitis, inflammatory bowel disease), malignancy, trauma, postsurgical conditions, portal hypertension, liver cirrhosis, hematological diseases, and acquired or inherited thrombophilia.[2,16,17]

Nonocclusive mesenteric ischemia (NOMI) is characterized by insufficient perfusion to the intestines without a marked occlusion in the visceral vessels. It is predisposed by another severe disease causing insufficient visceral perfusion either by, or as a combination of, diminished blood flow or increased abdominal pressure. Insufficient perfusion can be due to mesenteric vasoconstriction, hypovolemia, or cardiogenic low output. It is worth noting that, in the aforementioned situations, otherwise asymptomatic visceral artery stenosis may become symptomatic, blurring the distinction between Ao-AMI and NOMI.[2,18–20]

EPIDEMIOLOGY

In the general population, AMI of all etiologies is an uncommon disease. In a meta-analysis, the estimated pooled incidence was 6.2 per 100.000 persons per year, and variability between studies was 0.7 to 14.1/100.000/year.[21] In patients aged 75 years or older presenting to the emergency department with acute abdominal pain, the incidence of Ao-AMI is much higher (37.5/100.000/year). In fact, in that age group, it is more common than acute appendicitis or ruptured abdominal aortic aneurysm,[22] whereas, in patients younger than 50 years, the incidence of Ao-AMI is very low.[22,23] It is worth noting that not all AMI patients are diagnosed alive.[24] A recent study showed that nearly a third of Ao-AMI patients were diagnosed postmortem.[23] Previously, embolism was the leading cause of Ao-AMI, but in recent studies, thrombus has become equally or even more common.[5,10,22,23,25]

The incidence of VAMI has been estimated at 0.5 to 2.7/100.000/year.[22,26] In VAMI, patients are younger than in Ao-AMI, with age varying from 45 to 62 years in most cases.[17] Nonocclusive mesenteric ischemia incidence has been estimated to be 2.0/100.000/year.[18,22]

DIAGNOSIS

Clinical Presentation

The typical Ao-AMI patient is elderly and has a history of or risk factors for cardiovascular diseases. The symptoms may vary greatly. The only common feature in all patients is abdominal pain, commonly accompanied by nausea or vomiting and occasionally diarrhea, which can also include melena or hematochezia.[27] For EAMI, the onset of symptoms is typically very acute, and there may be an undiagnosed or untreated arrhythmia with synchronous emboli.[28] History of atherosclerosis or chronic mesenteric ischemia, that is, postprandial pain, diarrhea, and weight loss, may be present in AMI caused by thrombus. The symptoms of TAMI may develop suddenly or over multiple days or even weeks because of the presence of collateral circulation.[29]

Nonetheless, if the patient complains of highly intense pain in conjunction with a very soft abdomen on palpation ("pain out of proportion"), Ao-AMI should remain high in the differential diagnosis possibilities. Because of the variability of the symptoms, it is not uncommon that the diagnosis is suspected only after computed tomography (CT) imaging.[10,30] A high index of suspicion is crucial in diagnosing AMI.

Venous AMI patients usually present with more subtle pain and a longer history of symptoms. Mid-abdominal pain and predisposing factors may lead to suspecting the diagnosis. However, most diagnoses occur after obtaining an abdominal CT for abdominal complaints.[31]

Timely diagnosis of NOMI can be difficult unless it is caused by an abdominal compartment syndrome with appropriate intra-abdominal pressure measurement. A typical scenario involves a critically ill patient who develops an unexplained lactatemia in the setting of worsening organ dysfunctions. Predisposing clinical situations include post–cardiac surgery, aortic dissection, septic shock, and abdominal compartment syndrome.[20]

Laboratory Tests

Despite ongoing efforts to find a specific biomarker for AMI, none have yet proved helpful in clinical practice.[5,32–34] D-dimer was previously considered important in ruling out AMI.[2,35] This has been questioned since D-dimer and its behavior in different forms of AMI are largely unknown.[33] In addition, D-dimer can be elevated in most abdominal pathologies. Lactate

© 2025 Wolters Kluwer Health, Inc. All rights reserved.

Copyright © 2025 Wolters Kluwer Health, Inc. All rights reserved.

has low sensitivity to aid in specific diagnosis but does reflect systemic hypoperfusion and correlates with the risk of death irrespective of the underlying diagnosis.[36,37]

In sum, laboratory examinations do not help establish a specific diagnosis but may help in assessing disease severity. If the symptoms last more than 6 to 12 hours, leukocytes and C-reactive protein may be markedly elevated. When symptoms have an acute onset, diagnosis and management should not be delayed based on normal blood tests. Instead, there should be no hesitation in confirming the diagnosis and proceeding with revascularization to prevent the development of transmural bowel necrosis.

In VAMI, the etiology of venous thrombosis requires testing for inherited or acquired thrombophilia, especially if no clear risk factors exist. Common risk factors are malignancies, liver cirrhosis, recent trauma, pancreatitis, oral contraceptives, and inflammatory bowel disease.[2,38] The most common inherited factors are Factor V Leiden (9%) and prothrombin G20210A (7%) mutations, and the most common acquired factor was JAK2 V617F mutation (14%).[39,40] In addition, assessing systemic coagulation with specific laboratory tests in the acute phase may prove useful in monitoring the coagulation status and managing its disturbances.[38]

Imaging

In patients with suspected AMI of any etiology, a triple-phase computed tomography angiography (CTA) specified for AMI with a 1-mm maximum slice thickness should be obtained.[31,41] Computed tomography angiography is fast and accessible and has the highest sensitivity and specificity of all radiological examinations. Other imaging modalities do not have an established role in the acute setting.[2,3] Signs of intestinal ischemia may be subtle, difficult to identify, and it often underestimates the true extent of intestinal ischemia.[30,42–44] Concerns for contrast-induced nephropathy should not delay diagnostic imaging.[2,3] The radiology referral should mention AMI suspicion to enhance the accuracy of the radiology report.[29] All radiologists and clinicians are strongly encouraged to routinely evaluate mesenteric vessels in all abdominal CT imaging.[44]

On CTA, embolic occlusion often occurs in the proximal, noncalcified SMA just beyond the origin of the middle colic artery (Fig. 2*A*) but may be found in other locations as well.[45] Synchronous emboli are common.[44] Thrombotic occlusions occur frequently near the ostium at the root of the SMA/CA (Fig. 2*B*). Computed tomography angiography demonstrates calcified and noncalcified ostial plaques, thrombus, and often collateral arteries.[45] Mesenteric venous thrombosis is diagnosed by the presence of a contrast-filling defect and thrombosis in the associated veins.[46] In NOMI, as per definition, there are no marked changes within the mesenteric vessels. Occasionally, marked vasoconstriction is observed.

Bowel wall changes in AMI may present as decreased bowel enhancement and thinning, portomesenteric venous gas, and pneumatosis. More subtle changes are often seen, such as bowel wall thickening, mesenteric edema, free peritoneal fluid, and luminal dilatation.[47] All alterations have different diagnostic odds ratios (Table 1), and the most specific for transmural bowel necrosis were bowel wall thinning (98%) and portomesenteric venous gas (95%).[47] In VAMI, bowel and mesenteric edema with decreased enhancement are the typical findings of bowel ischemia.[46]

In clinical practice, left-sided colonic ischemia is not treated by revascularization. Bowel ischemia in the setting of

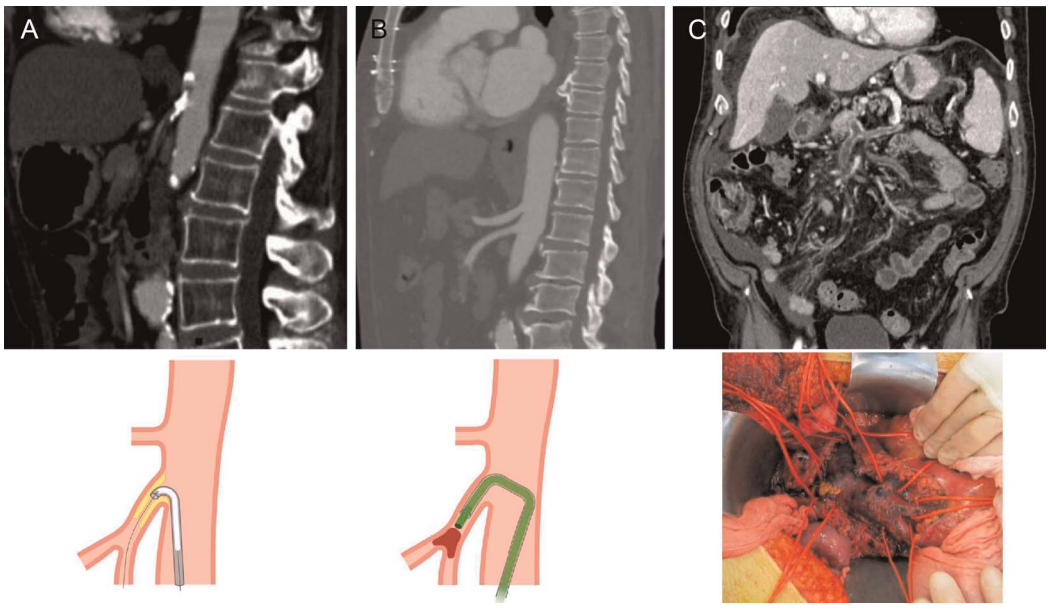

Figure 2. (*A–C*) Computed tomography angiography images of AMI with (*A*) SMA thrombus, (*B*) SMA embolism, and (*C*) vast venous occlusion of the portomesenteric system (CTA and open surgery). Endovascular treatment in image A may be challenging because of the acute angle and heavily calcified orifice of the SMA. The drawing also demonstrates how it may be difficult to navigate larger (covered) stents in acute angles, and an arterial dilatation is necessary to avoid stent dislodgement. Figure B demonstrates an embolus in an open SMA with a wide angle that should be easy to effectively be aspirated with the help of a guiding catheter.

© 2025 Wolters Kluwer Health, Inc. All rights reserved.

Copyright © 2025 Wolters Kluwer Health, Inc. All rights reserved.

TABLE 1. Predictive Value of CT Features for Predicting Transmural Intestinal Necrosis in AMI (Modified From Zeng et al., *European Radiology* (2023) 33:2792–2799)

CT Feature	Diagnostic Odds Ratio (95% Confidence Interval)	Specificity (%)	Sensitivity (%)
Bowel wall thinning	13.10 (3.71–46.25)	98	38
Pneumatosis intestinalis	5.78 (2.14–13.40)	N/A	N/A
Decreased or absent bowel wall enhancement	5.77 (2.95–11.30)	N/A	N/A
Portomesenteric venous gas	5.36 (2.14–13.40)	95	30
Bowel dilatation	3.23 (2.03–5.15)	N/A	N/A

small bowel strangulation due to adhesions or internal hernias is not a form of AMI, as the obstruction is external and not within the vessels, requiring surgical exploration with no role for revascularization. However, in literature, the nomenclature of what should be considered as AMI is not fully established.[48]

Predicting Transmural Bowel Necrosis

Several publications describe scoring systems to predict transmural bowel necrosis.[47,49,50] These scoring systems lack external validation, and none have been proven useful in clinical practice. Therefore, the decision to proceed to laparotomy needs to be made on an individual basis.

MANAGEMENT OF AO-AMI

Reversal of ischemia with prompt and effective revascularization is a prerequisite for the successful treatment of AMI. The method of revascularization is of secondary importance, and no current studies compare similar groups treated with open, hybrid, or endovascular techniques. Less invasive endovascular methods seem preferable whenever possible.[2,51] There has been a clear paradigm shift toward endovascular revascularization with evidence of improved survival.[10,52] Revascularization is almost always possible but does not guarantee a favorable outcome. Figure 3 depicts the different options for revascularization with increasing invasiveness. A hybrid operation room is preferred when laparotomy is considered, as this allows for the full spectrum of revascularization options.

Endovascular Treatment With Femoral or Brachial Artery Access

Percutaneous endovascular treatment is performed when there is no indication for laparotomy but may also be used during laparotomy. Both groin and (preferably left) upper arm access may be used. The angle between the aorta and SMA varies, and sagittal CTA images can identify potential problems when passing stents through tight angles and stenoses for example (Fig. 2). Guiding catheters may function well when the angle is wide and inserted using arm access, but large enough stents through acute angles are risky to position without sufficient support and predilatation. Therefore, in many cases, steerable sheets are a valid option.[2,53–55]

In TAMI and tight SMA stenosis, 6-mm and larger balloon expandable stents with high radial force should be used. Stents less than 6 mm are easier to navigate through tight angles but may have a higher risk of stent thrombosis. Covered versus uncovered stents is still an unsolved debate. A recent Dutch randomized controlled trial showed better long-term patency but no clinical benefit for covered stents. The covered stent group had more immediate problems because of the required larger sheath sizes and stent dislodgement. The relationship between stent size and long-term patency was not analyzed.[53] Another recent randomized "DISCOVER" trial showed no benefit in routinely using covered stents in the iliac arteries.[56]

An increasing number of aspiration devices and catheters exist.[57–59] Superior mesenteric artery is prone to dissection, and it may be necessary to aspirate distal branches of the artery. Therefore, gentle techniques and devices are important, and sometimes, catheters designed for intracerebral aspirations are helpful. Stable positioning may be achieved by guiding catheters further into the SMA or the more stable steerable sheets.[60,61] During aspiration, the disadvantage of nonremovable sheet valves may be solved with suitable guiding catheters or removable

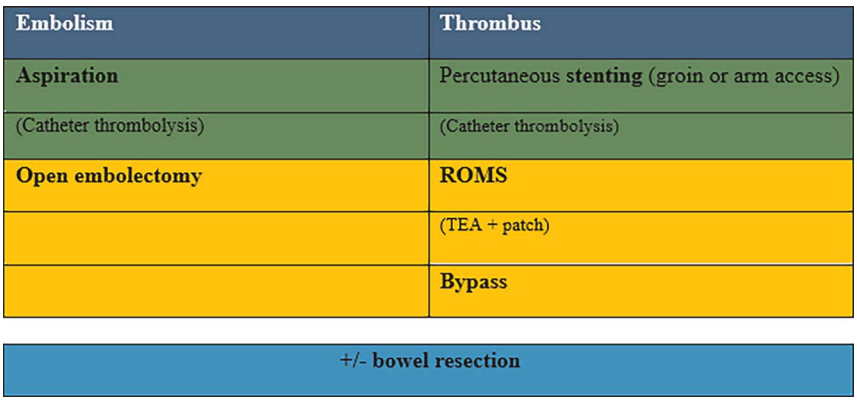

Figure 3. Options of revascularization. All options may be used in conjunction with a laparotomy, but the green options may be performed without open surgery. Our preference is to take all patients requiring open surgery to a hybrid operation room to keep all options available. The procedures in parenthesis are most often performed together with other techniques and the bolded ones are the most common. ROMS, retrograde open mesenteric stenting; TEA, thromboendarterectomy.

© 2025 Wolters Kluwer Health, Inc. All rights reserved.

Copyright © 2025 Wolters Kluwer Health, Inc. All rights reserved.

valves. Care should be taken to avoid distal embolism to smaller branches. Local administration of thrombolysis agents may be used but carries a risk of intestinal bleeding. We prefer on-table revascularization to longer thrombolysis.[62]

Completion angiogram is mandatory and may be combined with intraoperative high-frequency duplex ultrasound during laparotomy. Transit time flow measurement (TTFM) is a reliable tool during open surgery but requires SMA exploration, and we do not recommend exploration for TTFM purposes only. Transit time flow measurement flows >300 mL/min may be considered satisfactory but flows up to 1,000 mL/min or higher are possible in the SMA.[63] Additional angiographic views from different angles are advisable in low-flow situations, as significant stenoses may be missed in two-dimensional imaging.

Retrograde Open Mesenteric Stenting

Retrograde open mesenteric stenting during laparotomy has been previously described[64,65] and is our method of choice in many cases. We describe a simple and reliable technique, including "endo palpation" with an oversized balloon in Figure 4. The SMA should be surgically exposed, and a soft anterior arterial wall segment is almost always available for puncture. We prefer to use longer sheets, which make it possible to work away from the intestines and transition from smaller to larger guidewires and sheets, as described in the figure legend. After stenting, a Digital subtraction angiography (DSA) and TTFM are performed. The arterial puncture defect is closed, preferably with two interrupted transverse sutures. An endarterectomy and closure with a patch may occasionally be performed as an additional or stand-alone procedure.

Open Surgical Techniques

A traditional SMA embolectomy is a straightforward and effective procedure, especially when large emboli are already organized, carrying an increased risk of distal embolization during aspiration. Transverse arteriotomy is possible only in healthy arteries, and patch closures are often safe.[2,66,67]

Endovascular ante or retrograde stenting is often possible, but in cases with long calcified total occlusion, surgical bypass is still a valid and durable solution.[66] Prosthetic grafts should generally be avoided in a contaminated surgical field, but covering the prosthesis may avoid infection, especially when only small bowel is involved. Infection of these grafts is a major problem requiring high-risk reoperations. We always prefer venous conduits when feasible. Our preferred solution is to obtain inflow from the iliac arteries and use a vein graft in a wide loop to the SMA to avoid kinking. Transit time flow measurement flow measurement and mesenteric pulse palpation are suitable completion control methods. We do not perform completion angiography routinely in these cases.[2,68]

Bowel Management

Ischemic changes in the bowel begin in the mucosa. If timely revascularization is performed, ischemia may be reversed and progression to transmural necrosis may be avoided. The first decision is to determine whether the bowel needs to be assessed visually. The incidence of transmural bowel necrosis in Ao-AMI varies from 34% to 56%.[5,10,49,50] Therefore, laparotomy is not necessary in all cases and should be indicated selectively. There is no established clinical scoring system to reliably guide the decision-making process. The approach needs to be individualized. Clinical history should guide the management, including duration and severity of the symptoms, physical examination findings, presence of acute organ dysfunction, laboratory and imaging studies, frailty assessment, and success of the revascularization attempts. If transmural necrosis is suspected, operative intervention is indicated. Clinical peritonitis can be a late finding in AMI. Most surgeons perform laparotomy to assess the bowel.

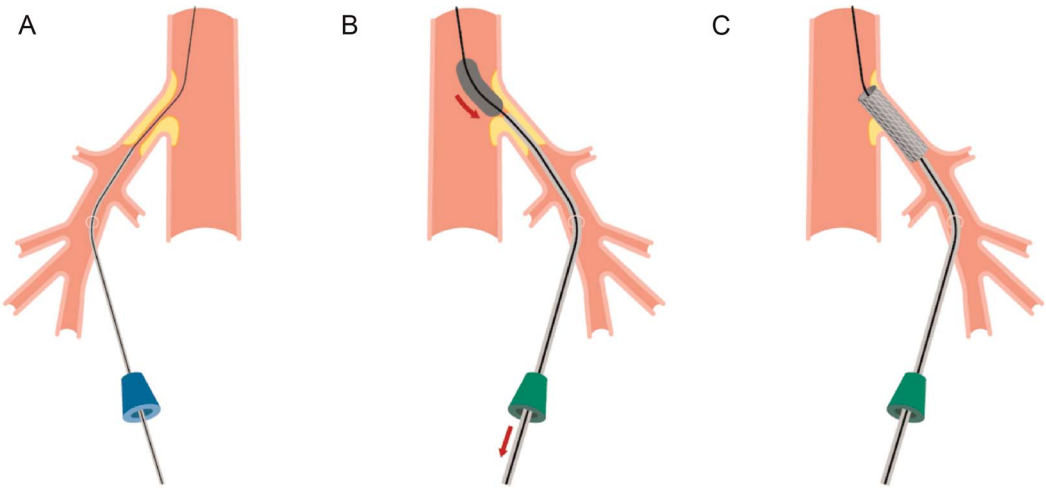

Figure 4. Retrograde open mesenteric stenting (ROMS). (*A*) After surgical exploration, a micropuncture is performed, a thin guidewire (e.g., 0.018″) is passed into the aorta, and the thin sheet may almost always follow the thin guidewire easily. (*B*) A RAO angle of >60° is often the best way to visualize the origin of the SMA. The thin wire is changed into a larger 0.035″ wire, and a larger 6- to 8-Fz sheet may be introduced. Predilatation is seldom needed. Long sheets are beneficial, as the procedure may be performed without touching the potentially contaminated intestine. An oversized balloon (e.g., 10 mm) is then inflated in the aorta and pulled back to be certain of the correct positioning ("endo palpation"). (*C*) Covered by the sheet, a bare metal or covered over the balloon-mounted stent is then expanded in the stenosis. Especially covered stents may be flared with the oversized balloon (or a larger one) in the aorta. It is important that the stent is large enough, typically 7 to 8 mm. RAO, right anterior oblique.

© 2025 Wolters Kluwer Health, Inc. All rights reserved.

Copyright © 2025 Wolters Kluwer Health, Inc. All rights reserved.

Bowel viability is assessed by color, bowel thickness, and peristalsis. Obvious transmural necrotic segments require resection. Resections are preferably performed after revascularization. In general, extensive resections of borderline viable bowel should be avoided in the index laparotomy.[2,3]

Indocyanine green fluorescence angiography is an experimental way to assess bowel viability intraoperatively.[69,70] While there are some promising reports of the use of indocyanine green, these methods are not yet validated and lack proof of influencing patient care and prognosis. Some surgeons perform laparoscopy instead of laparotomy.[71] This approach also lacks evidence to support its widespread use. Laparoscopy as a diagnostic tool, instead of a CTA, should be abandoned since early changes of bowel ischemia are not visible in the serosa. Concerns have been raised for the safety and efficacy of thoroughly assessing the full length of the intestinal segment affected by AMI. We advocate watchful waiting after endovascular revascularization instead of laparoscopy if the probability of transmural ischemia is low.

If an AMI patient has less than 100 cm (3 ft) of viable small bowel left, extensive small bowel resection is performed. If the patient survives the initial phase, the risk of developing short bowel syndrome with a long-term need for parenteral nutrition is very high.[72] Traditionally, these fragile patients have not been offered such vast bowel resections because of ethical reasons.[5] Short bowel syndrome with intestinal failure is primarily treated with parenteral nutrition, medications, and corrective surgery. A Finnish population-based study evaluated the potential for intestinal transplantation in AMI patients. Patients older than 70 years were excluded, and similar contraindications for solid organ transplants were used. It was estimated that 5% of patients younger than 70 years could have been potential candidates for intestinal transplantation.[73]

Role of a Staged Approach

Most guidelines advocate a staged approach for bowel resections.[2–4] In the first laparotomy, preferably after revascularization, transmural necrotic bowel segments are resected without anastomosis, and the abdomen is left open with a temporary abdominal closure device. After approximately 48 hours, the patient is taken back to the operating room for a second look laparotomy, which can be repeated if necessary. The bowel viability is assessed, additional necessary resections, anastomoses, or an ostomy are performed, and the abdomen is closed.

The benefits of multiple operations and an open abdomen strategy include a planned reassessment of the bowel and revascularization procedures, lower intra-abdominal pressure, and drainage of intra-abdominal fluids. However, routine open abdomen management in AMI patients lacks evidence. Open abdomen management does not come without disadvantages.[74] With modern, more restrictive fluid resuscitation strategies, abdominal compartment syndrome in Ao-AMI is practically nonexistent.[10,74,75]

Our strategy is to individualize the operative strategy. In a severely ill patient with acute onset of symptoms and a range of ischemic insults present at the index laparotomy, a staged approach is likely the best strategy. On the other end of the spectrum, a generally well-appearing patient with prolonged symptoms and a clearly demarcated necrotic bowel segment, a single-stage operation seems justifiable. The need for additional bowel resections after the index operation was 13% in an international multicenter study.[5]

The decision between performing a primary (delayed) anastomosis or an ostomy is not guided by the existing literature. We prefer primary anastomoses in our practice. Ostomies become an option in the case of anastomotic dehiscence. A recent international multicenter trial reported a 14% stoma rate at discharge.[5]

Medications

Once the diagnosis of Ao-AMI is established, there is a consensus to use broad-spectrum intravenous antibiotics.[2–4] There are very little solid scientific data for this practice. The rationale is to treat potential bacterial translocation from the damaged mucosa. In the rare case of bowel perforation, the antibiotics are continued to manage secondary peritonitis.[76] The duration of antibiotics is based on individual patient assessment; in our practice, duration of antibiotics is generally 1 to 7 days for most, but in the absence of bowel perforation, it may be shortened to 1 to 2 days. A small French study proposed that oral antibiotics may have a role in preventing the progression of ischemia into irreversible transmural necrosis.[77]

The use of vasoactive medications in the acute setting is complicated. For hypotensive patients, vasopressors decrease the volume of fluid resuscitation and thus reduce harmful fluid overload. On the other hand, vasopressors may cause visceral hypoperfusion by causing severe vasoconstriction, further worsening visceral ischemia. A systematic review concluded that there is a gap in the literature on the matter.[78]

Low-molecular-weight heparin (LMWH) or unfractionated heparin (UFH) should be used in the acute perioperative period. After the initial period, patients with arterial embolism need long-term anticoagulation, either with vitamin K antagonists or with direct oral anticoagulants. After stenting of the SMA, it is generally accepted that a short-term dual antiplatelet therapy (DAPT) is beneficial, but long-term DAPT has an increased bleeding risk compared with single antiplatelet therapy, and DAPT should be discontinued after 1 to 6 months.[79,80]

Postoperative Care

After revascularization and possible bowel resections, patients need to be monitored closely. The level of postoperative care is based on the severity of the disease and acute physiologic changes. Special attention should be paid to signs of anastomotic dehiscence, abdominal compartment syndrome, re-occlusion of the mesenteric arteries, or failure of the revascularization. Patients who have been managed without laparotomy should maintain a low threshold for surgical interventions if signs of transmural bowel necrosis develop.

Enteral feeding should be administered early once the need for possible vasoactive medications is low and bowel movements are present. This may prevent villi atrophy in the intestinal mucosa. Early high metabolic intake may increase mesenteric blood flow demand, so caution is advised.[3,81]

Malabsorption and short bowel syndrome in patients surviving the acute phase are rare. In a large Finnish cohort of 711 AMI patients, only two developed short bowel syndrome.[73] Patients with intestinal failure should receive specialized management and follow-up in multidisciplinary intestinal failure units.[82]

Follow-up

Patients who are at high risk for malabsorption but without the diagnosis of short bowel syndrome should be followed up and receive a dietarian consultation. Asymptomatic patients

© 2025 Wolters Kluwer Health, Inc. All rights reserved.

Copyright © 2025 Wolters Kluwer Health, Inc. All rights reserved.

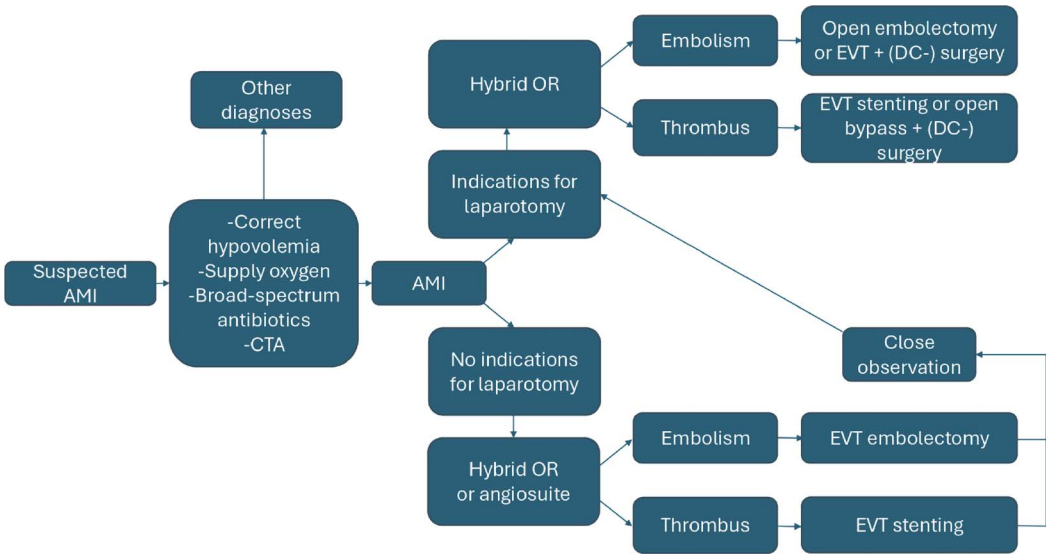

Figure 5. Acute mesenteric ischemia pathway (modified from Tolonen et al.[10]). EVT, endovascular therapy; DC, damage control.

have no indication for long-term surgical follow-up, and no definite recommendation for vascular follow-up can be given. It is possible to use ultrasound for the surveillance of SMA stenting.[83] Given the rarity of bypasses and the seriousness of occlusions, we perform an annual follow-up MRA in those patients and have treated some graft stenoses endovascularly. Patients with atherosclerotic disease should be treated according to general principles, including smoking cessation and antiplatelet and statin therapy.[79]

PATHWAY FOR AO-AMI

A pathway and care bundle for the management of Ao-AMI was published in 2021.[10] Implementing the pathway resulted in enhanced regional and in-hospital awareness, better diagnostics by contrast-enhanced CT imaging, fewer in-hospital delays, increased use of hybrid operating rooms, more decisive and effective revascularizations with increased use of endovascular therapy, refined perioperative care, and, hence, lower mortality. The 30-day mortality was lowered from 51% to 25%.[10] Figures 5 and 6 present the pathway. This pathway may be used as a template for other centers to examine and further develop local practices.

MANAGEMENT OF VAMI

The goals in the management of VAMI are preventing transmural bowel ischemia, enabling recanalization of the affected veins, preventing portal hypertension and its

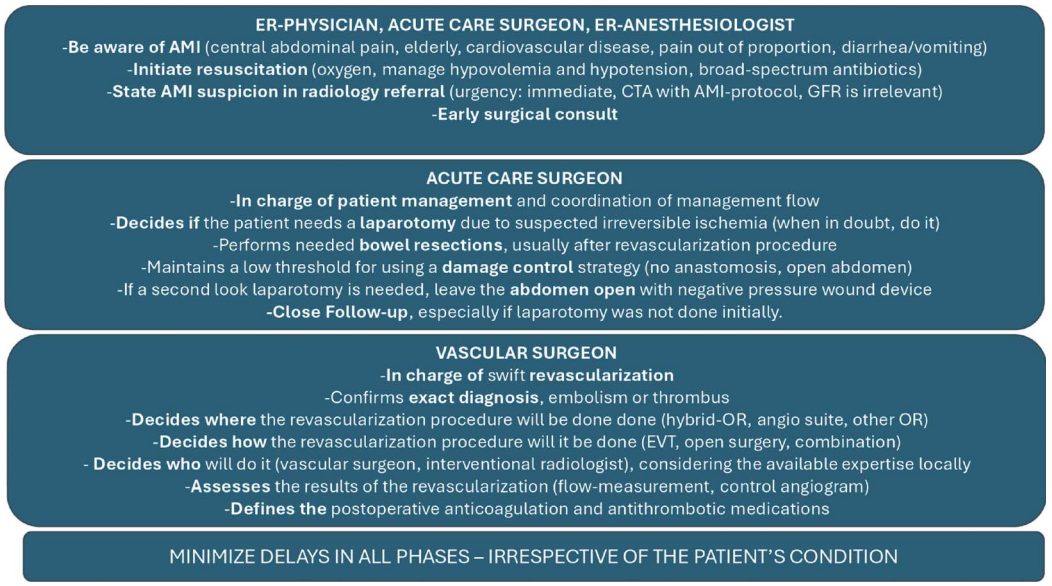
ER-PHYSICIAN, ACUTE CARE SURGEON, ER-ANESTHESIOLOGIST
-Be aware of AMI (central abdominal pain, elderly, cardiovascular disease, pain out of proportion, diarrhea/vomiting)
-Initiate resuscitation (oxygen, manage hypovolemia and hypotension, broad-spectrum antibiotics)
-State AMI suspicion in radiology referral (urgency: immediate, CTA with AMI-protocol, GFR is irrelevant)
-Early surgical consult
ACUTE CARE SURGEON
-In charge of patient management and coordination of management flow
-Decides if the patient needs a laparotomy due to suspected irreversible ischemia (when in doubt, do it)
-Performs needed bowel resections, usually after revascularization procedure
-Maintains a low threshold for using a damage control strategy (no anastomosis, open abdomen)
-If a second look laparotomy is needed, leave the abdomen open with negative pressure wound device
-Close Follow-up, especially if laparotomy was not done initially.
VASCULAR SURGEON
-In charge of swift revascularization
-Confirms exact diagnosis, embolism or thrombus
-Decides where the revascularization procedure will be done done (hybrid-OR, angio suite, other OR)
-Decides how the revascularization procedure will it be done (EVT, open surgery, combination)
- Decides who will do it (vascular surgeon, interventional radiologist), considering the available expertise locally
-Assesses the results of the revascularization (flow-measurement, control angiogram)
-Defines the postoperative anticoagulation and antithrombotic medications
MINIMIZE DELAYS IN ALL PHASES – IRRESPECTIVE OF THE PATIENT'S CONDITION

Figure 6. Acute mesenteric ischemia care bundle (modified from Tolonen et al.[10]). ER, emergency room; GFR, glomerular filtration rate; EVT, endovascular therapy.

© 2025 Wolters Kluwer Health, Inc. All rights reserved.

Copyright © 2025 Wolters Kluwer Health, Inc. All rights reserved.

complications, and thrombosis recurrence. Most patients do well with systemic anticoagulation. The early management after VAMI diagnosis is with LMWH or UFH. Definitive evidence regarding drug choice is lacking. In our practice, UFH is started for more severe cases until the symptoms improve, typically within 72 hours, and then we continue with LMWH. The advantage of UFH is that it can be quickly reversed by protamine if there is a need for interventions or profound bleeding. Usually, weeks after discharge from the hospital, anticoagulation can be changed to warfarin or direct oral anticoagulant. Supportive measures include correction of dehydration, nasogastric tube in ileus, nutritional support, and pain management.[17,84]

If symptoms persist or get worse, further measures need to be taken. Options include catheter thrombolysis either via SMA, with the idea that the mesenteric arterial and venous connections may be thrombosed, or with transhepatic techniques (direct puncture or transjugular/transhepatic).[17] It is also possible to perform a percutaneous transhepatic mechanical aspiration thrombectomy with the benefit of rapid thrombus removal. Evidence to support the choice and benefits of endovascular treatments is low, and the risk of bleeding exists.[17,85,86]

In the case of suspected transmural bowel necrosis, an emergency laparotomy is indicated, the necrotic bowel is resected, and open removal of a thrombus may be performed. Indications for operative exploration can be challenging. It is difficult to determine the proper length for bowel resection since the bowel can be quite swollen and discolored because of obstruction of the venous return. Bowel resection rates in VAMI have been reported between 16% and 29%.[5,17,84]

The etiology of the MVT needs to be clarified. The duration of anticoagulation should be at least 3 to 6 months if the underlying cause has been resolved. With persistent prothrombotic states or thrombophilia, the anticoagulation is lifelong. Patients need to be reimaged in an outpatient setting to evaluate possible recanalization of the affected veins. Recanalization is not always achieved, and venous collaterals usually develop.[87] The 1-year recanalization rates in a European multicenter study were 39% (13% initially) in the portal vein, 80% (57% initially) in the splenic vein, and 73% (42% initially) in the superior mesenteric vein.[88] In the case of portal hypertension, its complications, such as esophageal varices, should be sought and managed.[87]

MANAGEMENT OF NOMI

Prevention of NOMI is accomplished by maintaining adequate abdominal perfusion pressure. However, elevated vasopressor requirements are a well-known risk factor for NOMI.[89] When NOMI is identified, all measures should be taken to manage the underlying cause and prevent bowel necrosis. There are no well-established treatment protocols for NOMI. Patients may benefit from intra-arterial administration of vasodilators through a catheter placed in the SMA. No standards regarding which vasodilator to use or the ideal dosage exist.[90,91] A significant survival benefit with papaverine infusion was seen in a single study.[91] In a larger Japanese nationwide retrospective analysis with propensity matching, no treatment benefit could be seen in the vasodilator group.[92] Antibiotics should be considered because of the risk of bacterial translocation. When transmural bowel necrosis is suspected, a laparotomy with a staged approach and ad hoc bowel resections should be performed if the patient is considered to have a survivable prognosis. However, these situations carry a very high mortality rate.[3,20]

In the case of abdominal compartment syndrome, the diagnosis is usually easier to establish by measuring the intra-abdominal pressure. All conservative methods should be attempted to lower the intra-abdominal pressure and enhance the abdominal perfusion pressure. If these actions fail, a decompressive laparotomy should be ideally performed before the development of transmural bowel necrosis.[93]

In clinical practice, a common dilemma occurs in a previously asymptomatic patient with significant calcified narrowing, yet not complete occlusion of the SMA, who develops another severe condition with periods of low mean arterial pressure and possibly higher needs for visceral perfusion. The distinction between NOMI and TAMI may be difficult, but many of these patients would likely benefit from SMA stenting to enhance visceral perfusion.[18]

PROGNOSIS

In Ao-AMI, the reported mortality rates strongly depend on the study cohort. Without active attempts for revascularization in thrombotic arterial occlusion, the mortality is approximately 80%.[1] In some patients, active care is withdrawn because of vast necrosis or presumed futility of care. A population-based study from Finland showed that nearly a third of patients are diagnosed postmortem.[73] The lowest mortality rates are reported in subgroup studies of selected and transferred patients into specialized units.[49,94] In unselected patients, the mortality is usually reported at around 50%[3,5] and lowest at 25% with specific management pathways.[10] There are a variety of independent risk factors associated with mortality, the most important being preoperative acute organ dysfunctions, chronic diseases with poor functional capacity, lactate levels, and delays in management.[10,95,96] It has been shown that AMI patients who survive the initial phase have a reasonable prognosis, with over 80% of patients being alive after 1 year and with a low incidence of permanent institutionalization.[22,97]

In VAMI, mortality is significant, around 30% in historical studies.[21] In more recent studies, mortality rates in MVT have been reported between 3% and 10%, which may better reflect the current prognosis with contemporary management.[17,84] In NOMI, mortality is the highest, cited as 50% to 80%.[1,21]

CHALLENGES AND FUTURE DIRECTIONS

Acute mesenteric ischemia may well be the most lethal diagnosis in emergency general surgery. However, there is plenty of room for improvement, especially in the management of Ao-AMI. Acute mesenteric ischemia is not a rarity in the elderly population with comorbidities, and those who survive the initial hospitalization can live a meaningful life.[22,97] There remain several challenges to improve, which include a lack of general knowledge of the disease, attitudes of medical personnel, variability in symptoms, missed or delayed diagnoses, accessibility to optimal facilities, management delays, selecting the proper individualized management pathway, and availability of effective revascularization by skilled teams.

However, these challenges can be tackled by having a high index of suspicion for AMI, raising regional awareness, and

© 2025 Wolters Kluwer Health, Inc. All rights reserved.

Copyright © 2025 Wolters Kluwer Health, Inc. All rights reserved.

streamlining local multidisciplinary pathways. Emergent and effective revascularization is of paramount importance in Ao-AMI. Management of AMI requires nuance, and not all patients will require a laparotomy, just as not all bowel ischemia is mesenteric ischemia. Nonetheless, AMI patients can be managed in any well-equipped hospital willing to provide high-quality emergency general surgery care. Future studies should focus on existing literature gaps and challenges, such as enhancing diagnostics and early detection of transmural bowel necrosis. The role of artificial intelligence and machine learning in AMI is in its infancy but may prove to be an asset in the future.[20,98–100]

AUTHORSHIP

Both authors contributed to literature search, study design, writing, and critical revision of the manuscript.

ACKNOWLEDGMENTS

We express our gratitude to Mr. Petteri Mattila for providing the illustrations in the manuscript.

DISCLOSURE

Conflicts of Interest: Author Disclosure forms have been supplied and are provided as Supplemental Digital Content (http://links.lww.com/TA/E299).

REFERENCES

1. Schoots, IG, Koffeman, GI, Legemate, DA, Levi, M, van Gulik, TM. Systematic review of survival after acute mesenteric ischaemia according to disease aetiology. *Br J Surg* 2003;91:17–27.
2. Björck M, Koelemay M, Acosta S, Bastos Goncalves F, Kölbel T, Kolkman JJ, et al. Editor's choice — Management of the diseases of mesenteric arteries and veins: clinical practice guidelines of the European Society of Vascular Surgery (ESVS). *Eur J Endovasc Surg*. 2017;53:460–510.
3. Bala M, Catena F, Kashuk J, De Simone B, Augusto Gomes C, Weber D, et al. Acute mesenteric ischemia: updated guidelines of the World Society of Emergency Surgery. *World J Emerg Surg*. 2022;17:54.
4. Tilsed JVT, Casamassaima A, Kurihara H, Mairani D, Martinez I, Pereira J, et al. 253-270 ESTES guidelines: acute mesenteric ischaemia. *Eur J Trauma Emerg Surg*. 2016;42:42–270.
5. Blaser AR, Mändul M, Björck M, Acosta S, Bala M, Bodnar Z, et al. Incidence, diagnosis, management and outcome of acute mesenteric ischaemia: a prospective, multicentre observational study (AMESI study). *Crit Care*. 2014;28:32.
6. Blauw J, Bulut T, Eenhoorn P, Beuk RJ, Brusse-Keizer M, Kolkman JJ, et al. Chronic mesenteric ischemia: when and how to intervene on patients with celiac/SMA stenosis. *J Cardiovasc Surg (Torino)*. 2017;58:321–328.
7. Lemma AN, Tolonen M, Vikatmaa P, Mentula P, Vikatmaa L, Kantonen I, et al. Choice of first emergency room affects the fate of patients with acute mesenteric ischaemia: the importance of referral patterns and triage. *Eur J Vasc Endovasc Surg*. 2019;57:842–849.
8. Zettervall SL, Lo RC, Soden PA, Deery SE, Ultee KH, Pinto DS, et al. Trends in treatment and mortality for mesenteric ischemia in the United States from 2000 to 2012. *Ann Vasc Surg*. 2017;42:111–119.
9. Hess B, Cahenzli M, Forbes A, Burgos R, Coccolini F, Corcos O, et al. Management of acute mesenteric ischaemia: results of a worldwide survey. *Clin Nutrition Espen*. 2023;54:194–205.
10. Tolonen M, Lemma A, Vikatmaa P, Peltola E, Mentula P, Björkman P, et al. The implementation of a pathway and care bundle for the management of acute occlusive arterial mesenteric ischemia reduced mortality. *J Trauma Acute Care*. 2021;91:480–488.
11. Garzelli L, Abdallah IB, Nuzzo A, Zappa M, Corcos O, Burgio MD, et al. Insights into acute mesenteric ischaemia: an up-to-date, evidence-based review from a mesenteric stroke centre unit. *Br J Radiol*. 2023;96:20230232.
12. Jeays AD, Lawford PV, Gillott R, Spencer PA, Bardhan KD, Hose DR. A framework for the modeling of gut blood flow regulation and postprandial hyperaemia. *World J Gastroenterol*. 2007;13:1393–1398.
13. Gatt M, MacFie J, Anderson ADG, Howell G, Reddy BS, Suppiah A, et al. Changes in superior mesenteric artery blood flow after oral, enteral, and parenteral feeding in humans*. *Crit Care Med*. 2009;37:171–176.
14. Blauw JTM, Metz FM, Brusse-Keizer M, Rijnja P, Bruno MJ, Geelkerken RH, et al. Coeliac artery release or sham operation in patients suspected of having median arcuate ligament syndrome: the CARoSO study. *Eur J Vasc Endovasc Surg*. 2022;64:573–574.
15. Harjola PT. A rare obstruction of the coeliac artery. Report of a case. *Ann Chir Gynaecol Fenn*. 1963;52:547–550.
16. DeLeve LD, Valla D-C, Garcia-Tsao G. American Association for the Study Liver Diseases. Vascular disorders of the liver. *Hepatology*. 2009; 49:1729–1764.
17. Acosta S, Salim S. Management of acute mesenteric venous thrombosis: a systematic review of contemporary studies. *Scand J Surg*. 2020;170: 145749692096907–145749692096908.
18. Acosta S, Ögren M, Sternby N-H, Bergqvist D, Björck M. Fatal nonocclusive mesenteric ischaemia: population-based incidence and risk factors. *J Intern Med*. 2006;259:305–313.
19. Bala M, Kashuk J, Moore EE, Kluger Y, Biffl W, Gomes CA, et al. Acute mesenteric ischemia: guidelines of the World Society of Emergency Surgery. *World J Emerg Surg*. 2017;7(12):38.
20. Bourcier S, Klug J, Nguyen LS. Non-occlusive mesenteric ischemia: diagnostic challenges and perspectives in the era of artificial intelligence. *World J Gastroenterol*. 2021;27:4088–4103.
21. Tamme K, Blaser AR, Laisaar KT, Mändul M, Kals J, Forbes A, et al. Incidence and outcomes of acute mesenteric ischaemia: a systematic review and meta-analysis. *BMJ Open*. 2022;12:e062846.
22. Kärkkäinen JM, Lehtimäki TT, Manninen H, Paajanen H. Acute mesenteric ischemia is a more common cause than expected of acute abdomen in the elderly. *J Gastrointest Surg*. 2015;19:1407–1414.
23. Lemma A, Tolonen M, Vikatmaa P, Mentula P, Kantonen I, But A, et al. Epidemiology, diagnostics and outcomes of acute occlusive arterial mesenteric ischaemia — a population-based study. *Eur J Vasc Endovasc*. 2022;64: 646–653.
24. Acosta S, Ögren M, Sternby NH, Bergqvist D, Björck M. Incidence of acute Thrombo-embolic occlusion of the superior mesenteric artery—a population-based study. *Eur J Vasc Endovasc Surg*. 2004;27:145–150.
25. Blaser AR, Starkopf J, Björck M, Forbes A, Kase K, Kiisk E, et al. Diagnostic accuracy of biomarkers to detect acute mesenteric ischaemia in adult patients: a systematic review and meta-analysis. *World J Emerg Surg*. 2023; 18:44.
26. Acosta S. Epidemiology of mesenteric vascular disease: clinical implications. *Semin Vasc Surg*. 2010;23:4–8.
27. Park WM, Gloviczki P, Cherry KJ Jr., Hallett JW Jr., Bower TC, Panneton JM, et al. Contemporary management of acute mesenteric ischemia: factors associated with survival. *J Vasc Surg*. 2002;35:445–452.
28. Acosta S, Björck M. Acute thrombo-embolic occlusion of the superior mesenteric artery: a prospective study in a well defined population. *Eur J Vasc Endovasc Surg*. 2003;26:179–183.
29. Kärkkäinen JM, Lehtimäki TT, Saari P, Hartikainen J, Rantanen T, Paajanen H, et al. Endovascular therapy as a primary revascularization modality in acute mesenteric ischemia. *Cardiovasc Interv Radiol*. 2015;38:1119–1129.
30. Lehtimäki TT, Kärkkäinen JM, Saari P, Manninen H, Paajanen H, Vanninen R. Detecting acute mesenteric ischemia in CT of the acute abdomen is dependent on clinical suspicion: review of 95 consecutive patients. *Eur J Radiol*. 2015;84:2444–2453.
31. Salim S, Ekberg O, Elf J, Zarrouk M, Gottsäter A, Acosta S. Clinical implications of CT findings in mesenteric venous thrombosis at admission. *Emerg Radiol*. 2018;25:407–413.
32. Khan SM, Emile SH, Wang Z, Agha MA. Diagnostic accuracy of hematological parameters in acute mesenteric ischemia-a systematic review. *Int J Surg*. 2019;66:18–27.
33. Blauw JTM, Metz FM, Nuzzo A, van Etten-Jamaludin FS, Brusse-Keiser M, Boermeester MA, et al. The diagnostic value of biomarkers in acute mesenteric ischaemia is insufficiently substantiated: a systematic review. *Eur J Vasc Endovasc Surg*. 2024;67:554–569.
34. Tamme K, Acosta S, Biloslavo A, Björck M, Casian D, Damaskos D, et al. Biomarkers in prediction of acute mesenteric Ischaemia: a prospective multicentre study (BIPAMI study): a study protocol. *BMC Surg*. 2024; 24(1):201.
35. Akyildiz H, Akcan A, Oztürk A, Sozuer E, Kucuk C, Karahan I. The correlation of the D-dimer test and biphasic computed tomography with mesenteric computed tomography angiography in the diagnosis of acute mesenteric ischemia. *Am J Surg*. 2009;197:429–433.

© 2025 Wolters Kluwer Health, Inc. All rights reserved.

Copyright © 2025 Wolters Kluwer Health, Inc. All rights reserved.

36. Studer P, Vaucher A, Candinas D, Schnüriger B. The value of serial serum lactate measurements in predicting the extent of ischemic bowel and outcome of patients suffering acute mesenteric ischemia. *J Gastrointest Surg*. 2015;19:751–755.
37. Singer M, Deutschman CS, Seymour CW, Shankar-Hari M, Annane D, Bauer M, et al. The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). *JAMA*. 2016;23(315):801.
38. Middeldorp S, Nieuwlaat R, Baumann Kreuziger L, Coppens M, Houghton D, James AH, et al. American Society of Hematology 2023 guidelines for management of venous thromboembolism: thrombophilia testing. *Blood Adv* 2023;7:7101–38.
39. Zarrouk M, Salim S, Elf J, Gottsäter A, Acosta S. Testing for thrombophilia in mesenteric venous thrombosis — retrospective original study and systematic review. *Best Pr Res Clin Gastroenterol*. 2017;31:39–48.
40. Ortel TL, Neumann I, Ageno W, Beyth R, Clark NP, Cuker A, et al. American Society of Hematology 2020 guidelines for management of venous thromboembolism: treatment of deep vein thrombosis and pulmonary embolism. *Blood Adv*. 2020;4:4693–4738.
41. Menke J. Diagnostic accuracy of multidetector CT in acute mesenteric ischemia: systematic review and meta-analysis. *Radiology*. 2010;256:93–101.
42. Copin P, Ronot M, Nuzzo A, Maggiori L, Bouhnik Y, Corcos O, et al. Inter-reader agreement of CT features of acute mesenteric ischemia. *Eur J Radiol*. 2018;105:87–95.
43. Olson MC, Bach CR, Wells ML, Andrews JC, Khandelwal A, Welle CL, et al. Imaging of bowel ischemia: an update, from the AJR special series on emergency radiology. *Am J Roentgenol*. 2023;220:173–185.
44. Wadman M, Block T, Ekberg O, Syk I, Elmståhl S, Acosta S. Impact of MDCT with intravenous contrast on the survival in patients with acute superior mesenteric artery occlusion. *Emerg Radiol*. 2010;17:171–178.
45. Hagspiel KD, Flors L, Hanley M, Norton PT. Computed tomography angiography and magnetic resonance angiography imaging of the mesenteric vasculature. *Tech Vasc Interv Radiol*. 2015;18:2–13.
46. Henes FO, Pickhardt PJ, Herzyk A, Lee SJ, Motosugi U, Derlin T, et al. CT angiography in the setting of suspected acute mesenteric ischemia: prevalence of ischemic and alternative diagnoses. *Abdom Radiol*. 2017;42:1152–1161.
47. Zeng Y, Yang F, Hu X, Zhu F, Chen W, Lin W. Radiological predictive factors of transmural intestinal necrosis in acute mesenteric ischemia: systematic review and meta-analysis. *Eur Radiol*. 2023;33:2792–2799.
48. Acosta S, Coopersmith CM, Blaser AR. Nomenclature issues: non-occlusive mesenteric ischaemia and colon ischaemia. *Intensiv Care Med*. 2024;50:1–2.
49. Nuzzo A, Maggiori L, Ronot M, Becq A, Plessier A, Gault N, et al. Predictive factors of intestinal necrosis in acute mesenteric ischemia: prospective study from an intestinal stroke center. *Am J Gastroenterol*. 2017;112:597–605.
50. Emile SH, Khan SM, Barsoum SH. Predictors of bowel necrosis in patients with acute mesenteric ischemia: systematic review and meta-analysis. *Updates Surg*. 2021;73:47–57.
51. Andraska E, Haga L, Li X, Avgerinos E, Singh M, Chaer R, et al. Retrograde open mesenteric stenting should be considered as the initial approach to acute mesenteric ischemia. *J Vasc Surg*. 2020;72:1260–1268.
52. Sakamoto T, Kubota T, Funakoshi H, Lefor AK. Multidisciplinary management of acute mesenteric ischemia: surgery and endovascular intervention. *World J Gastrointest Surg*. 2021;13:806–813.
53. Terlouw LG, van Dijk LJD, Noord D van, Bakker OJ, Bijdevaate DC, Erler NS, et al. Covered versus bare-metal stenting of the mesenteric arteries in patients with chronic mesenteric ischaemia (CoBaGI): a multicentre, patient-blinded and investigator-blinded, randomised controlled trial. *Lancet Gastroenterol Hepatol* 2024;9:299–309.
54. Oderich GS, Tallarita T, Gloviczki P, Duncan AA, Kalra M, Misra S, et al. Mesenteric artery complications during angioplasty and stent placement for atherosclerotic chronic mesenteric ischemia. *J Vasc Surg*. 2012;55:1063–1071.
55. Oderich GS, Erdoes LS, LeSar C, Mendes BC, Gloviczki P, Cha S, et al. Comparison of covered stents versus bare metal stents for treatment of chronic atherosclerotic mesenteric arterial disease. *J Vasc Surg*. 2013;58:1316–1324.
56. Bekken JA, Vroegindeweij D, Vos JA, de Vries JPM, Lardenoije JWHP, Petri BJ, et al. Editor's choice — two year results of the randomised DISCOVER trial comparing covered versus bare metal stents in the common iliac artery. *Eur J Vasc Endovasc Surg* 2023;65:359–68.
57. Xu R, Tang L, Qin S, Zhang S, Wang M. Percutaneous mechanical thrombectomy using the AcoStream thrombus aspiration system for acute superior mesenteric artery embolism. *Ann Vasc Surg*. 2024;106:264–272.
58. Thurner A, Peter D, Dalla Torre G, Flemming S, Kickuth R. Safety, efficacy and outcome of rotational thrombectomy assisted endovascular revascularisation of the superior mesenteric artery in acute thromboembolic mesenteric ischaemia. *RöFo - Fortschritte auf dem Geb Röntgenstrahlen Bild Verfahr*. 2024;196:1055–1062.
59. Kawano M, Azuma M, Hattori Y, Ikushima I, Kai K, Higuchi K, et al. Short-term experience with aspiration embolectomy using a ACE68 reperfusion catheter to treat embolic superior mesenteric artery occlusion. *Emerg Radiol*. 2021;28:675–678.
60. Liu YR, Tong Z, Hou CB, Cui SJ, Guo LR, Qi YX, et al. Aspiration therapy for acute embolic occlusion of the superior mesenteric artery. *World J Gastroenterol*. 2019;25:848–858.
61. Choi KS, Kim JD, Kim HC, Min SI, Min SK, Jae HJ, et al. Percutaneous aspiration embolectomy using guiding catheter for the superior mesenteric artery embolism. *Korean J Radiol*. 2015;16:736–743.
62. Garzelli L, Paulatto L, Corcos O, Castier Y, Ronot M, Abdallah IB. Catheter-directed thrombolysis using recombinant tissue plasminogen activator for the treatment of isolated mesenteric artery dissection with acute mesenteric ischemia. *Diagn Interv Imaging*. 2023;104:384–385.
63. Vikatmaa P, Albäck A. Intraoperative quality control with transit time flow measurement. *Gefässchirurgie*. 2018;23(8):580–585.
64. Milner R, Woo EY, Carpenter JP. Superior mesenteric artery angioplasty and stenting via a retrograde approach in a patient with bowel ischemia. *Vasc Endovascular Surg*. 2004;38:89–91.
65. Pisimisis GT, Oderich GS. Technique of hybrid retrograde superior mesenteric artery stent placement for acute-on-chronic mesenteric ischemia. *Ann Vasc Surg*. 2011;25:132.e7–132.e11.
66. Goto D, Yanishi K, Ozawa T, Yoshimura J, Kawamata H, Fujioka A, et al. Comparison of endovascular therapy and open surgical revascularization in patients with acute superior mesenteric artery occlusion: a large-scale analysis based on the JROAD-DPC database. *J Am Hear Assoc: Cardiovasc Cerebrovasc Dis*. 2024;13:e035017.
67. Sise MJ. Mesenteric ischemia: the whole Spectrum. *Scand J Surg*. 2010;99(2):106–110.
68. Kärkkäinen JM, Acosta S. Acute mesenteric ischemia (part II) — vascular and endovascular surgical approaches. *Best Pract Res Clin Gastroenterol*. 2017;1(31):27–38.
69. Karampinis I, Keese M, Jakob J, Stasiunaitis V, Gerken A, Attenberger U, et al. Indocyanine green tissue angiography can reduce extended bowel resections in acute mesenteric ischemia. *J Gastrointest Surg*. 2018;22:2117–2124.
70. Joosten JJ, Longchamp G, Khan MF, Lameris W, van Berge Henegouwen MI, Bemelman WA, et al. The use of fluorescence angiography to assess bowel viability in the acute setting: an international, multi-centre case series. *Surg Endosc* 2022;36:7369–75.
71. Gonenc M, Dural CA, Kocatas A, Buyukasik S, Karabulut M, Alis H. The impact of early diagnostic laparoscopy on the prognosis of patients with suspected acute mesenteric ischemia. *Eur J Trauma Emerg Surg*. 2013;39:185–189.
72. Messing B, Crenn P, Beau P, Boutron-Ruault MC, Rambaud JC, Matuchansky C. Long-term survival and parenteral nutrition dependence in adult patients with the short bowel syndrome. *Gastroenterology*. 1999;117:1043–1050.
73. Lemma A, Pikkarainen S, Pohju A, Tolonen M, Mentula P, Vikatmaa P, et al. Potential for intestinal transplantation after acute mesenteric ischemia in patients aged less than 70 years: a population-based study. *Scand J Surg*. 2023;112:77–85.
74. Proaño-Zamudio JA, Argandykov D, Renne A, Gebran A, Dorken-Gallastegi A, Paranjape CN, et al. Revisiting abdominal closure in mesenteric ischemia: is there an association with outcome? *Eur J Trauma Emerg Surg*. 2023;49:2017–2024.
75. Acosta S, Kärkkäinen J. Open abdomen in acute mesenteric ischemia. *Anaesthesiol Intensive Ther*. 2019;51:159–162.
76. Tian Y, Dhara S, Barrett CD, Richman AP, Brahmbhatt TS. Antibiotic use in acute mesenteric ischemia: a review of the evidence and call to action. *Thromb J*. 2023;21:39.
77. Nuzzo A, Maggiori L, Paugam-Burtz C, Cazals-Hatem D, Ronot M, Huguet A, et al. Oral antibiotics reduce intestinal necrosis in acute mesenteric ischemia: a prospective cohort study. *Am J Gastroenterol*. 2018;114:1–5.

© 2025 Wolters Kluwer Health, Inc. All rights reserved.

Copyright © 2025 Wolters Kluwer Health, Inc. All rights reserved.

78. Brennan CA, Osei-Bonsu P, McClenaghan RE, Nassar A, Forget P, Kaye C, et al. Vasoactive agents in acute mesenteric ischaemia in critical care. A systematic review. *F1000Research*. 2021;10:453.
79. Twine CP, Kakkos SK, Aboyans V, Baumgartner I, Behrendt CA, Bellmunt-Montoya S, et al. Editor's choice — European Society for Vascular Surgery (ESVS) 2023 clinical practice guidelines on antithrombotic therapy for vascular diseases. *Eur J Vasc Endovasc Surg*. 2023;65:627–689.
80. Nana P, Koelemay MJW, Leone N, Brodis A, van den Berg JC, de Bruin JL, et al. A systematic review of endovascular repair outcomes in atherosclerotic chronic mesenteric ischaemia. *Eur J Vasc Endovasc Surg*. 2023;66:632–643.
81. Yang S, Guo J, Ni Q, Chen J, Guo X, Xue G, et al. Enteral nutrition improves clinical outcome and reduces costs of acute mesenteric ischaemia after recanalisation in the intensive care unit. *Clin Nutr*. 2019;38:398–406.
82. Pironi L, Cuerda C, Jeppesen PB, Joly F, Jonkers C, Krznarić Ž, et al. ESPEN guideline on chronic intestinal failure in adults — update 2023. *Clin Nutr*. 2023;42:1940–2021.
83. Baker AC, Chew V, Li CS, Lin TC, Dawson DL, Pevec WC, et al. Application of duplex ultrasound imaging in determining in-stent stenosis during surveillance after mesenteric artery revascularization. *J Vasc Surg*. 2012; 56:1364–1371.
84. Salim S, Zarrouk M, Elf J, Gottsäter A, Ekberg O, Acosta S. Improved prognosis and low failure rate with anticoagulation as first-line therapy in mesenteric venous thrombosis. *World J Surg*. 2018;42:3803–3811.
85. Gao F, Wang L, Pan J, Yin Y, Wang J, Xu X, et al. Efficacy and safety of thrombolytic therapy for portal venous system thrombosis: a systematic review and meta-analysis. *J Intern Med*. 2023;293:212–227.
86. Wang L, Wang E, Liu F, Zhang W, Shu X, Guo D, et al. A systematic review and meta-analysis on endovascular treatment as an attractive alternative for acute superior mesenteric venous thrombosis. *Vascular*. 2022;30:331–340.
87. Lemma A, Åberg F, Mäkisalo H, Vikatmaa P, Mentula P, Leppäniemi A, et al. Predictors of insufficient recanalization and portal hypertensive complications after treatment of non-cirrhotic, non-malignant portal vein thrombosis — a population-based study. *Scand J Gastroenterol*. 2020;55:1324–1332.
88. Plessier A, Darwish-Murad S, Hernandez-Guerra M, Consigny Y, Fabris F, Trebicka J, et al. Acute portal vein thrombosis unrelated to cirrhosis: a prospective multicenter follow-up study. *Hepatology*. 2010;51:210–218.
89. Lim JY, Kim JB, Jung SH, Choo SJ, Chung CH, Lee JW. Risk factor analysis for nonocclusive mesenteric ischemia following cardiac surgery. *Medicine*. 2017;96:e8029.
90. Stahl K, Rittgerodt N, Busch M, Maschke SK, Schneider A, Manns MP, et al. Nonocclusive mesenteric ischemia and interventional local vasodilatory therapy: a meta-analysis and systematic review of the literature. *J Intensiv Care Med*. 2020;35:128–139.
91. Winzer R, Fedders D, Backes M, Ittermann T, Gründling M, Mensel B, et al. Local intra-arterial vasodilator infusion in non-occlusive mesenteric ischemia significantly increases survival rate. *Cardiovasc Interv Radiol*. 2020;43:1148–1155.
92. Takiguchi T, Nakajima M, Ohbe H, Sasabuchi Y, Tagami T, Kaszynski RH, et al. Association between postoperative adjuvant vasodilator therapy and in-hospital mortality for non-occlusive mesenteric ischemia: a nationwide observational study. *J Nippon Méd Sch*. 2024;91:316–321.
93. Kirkpatrick AW, Roberts DJ, Waele J, Jaeschke R, Malbrain ML, de Keulenaer B, et al. Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome. *Intensive Care Med* 2013;39:1190–1206
94. Najdawi M, Garzelli L, Nuzzo A, Huguet A, Raynaud L, Paulatto L, et al. Endovascular revascularization of acute arterial mesenteric ischemia: report of a 3-year experience from an intestinal stroke center unit. *Eur Radiol*. 2022;32:5606–5615.
95. Otto CC, Czigany Z, Heise D, Bruners P, Kotelis D, Lang SA, et al. Prognostic factors for mortality in acute mesenteric ischemia. *J Clin Med*. 2022;11:3619.
96. Sumbal R, Baig MMA, Sumbal A. Predictors of mortality in acute mesenteric ischemia: a systematic review and meta-analysis. *J Surg Res*. 2022; 275:72–86.
97. Pengermä P, Venesmaa S, Karjalainen J, Ukkonen M, Saari P, Kärkkäinen JM. Long-term outcome after implementation of endovascular-first strategy to treat acute mesenteric ischemia. *J Vasc Surg*. 2023;78:1524–1530.
98. Strand-Amundsen RJ, Tronstad C, Reims HM, Reinholt FP, Høgetveit JO, Tønnessen TI. Machine learning for intraoperative prediction of viability in ischemic small intestine. *Physiol Meas*. 2018;39:105011.
99. Jiang M, Li CL, Pan CQ, Lv WZ, Ren YF, Cui XW, et al. Nomogram for predicting transmural bowel infarction in patients with acute superior mesenteric venous thrombosis. *World J Gastroenterol*. 2020;26:3800–3813.
100. Harmantepe AT, Dulger UC, Gonullu E, Dikicier E, Þentürk A, Eröz E. A method for predicting mortality in acute mesenteric ischemia: machine learning. *Turk J Trauma Emerg Surg*. 2024;30:487–492.

© 2025 Wolters Kluwer Health, Inc. All rights reserved.

Copyright © 2025 Wolters Kluwer Health, Inc. All rights reserved.